

Alternaria leaf blight of wheat -*Alternaria triticina*

Alternaria triticina is the causal agent of *Alternaria* leaf blight of wheat, initially described from India. It can cause major damage to susceptible wheat varieties under wet or humid conditions especially on susceptible cultivars of durum and bread wheat. This is one of several species in the genus that have been isolated from wheat leaves; it is demonstrated to be pathogenic, while others appear to be primarily saprophytes. The species has been reported from other hosts and other countries on several continents, but recent taxonomic examinations (Simmons, 2007; Mercado Vergnes et al., 2006) have only supported its presence in southern and southwestern Asia. It is difficult to identify easily by morphological examination. Major wheat-growing countries may be considered at risk if seed will be imported, particularly from South Asia. It is definitely seedborne, so the possibility of accidental introduction is a threat for growers using imported seed.

Alternaria triticina Prasada & Prabhu, 1962

Mycelium initially hyaline, becoming olive-buff to deep olive-buff, branched, septate, 2-7 µm wide. Conidiophores similar to mycelium in colour, septate, unbranched or occasionally branched, erect, broader towards the distal end, on the host single or fasciculate, emerging through stomata, amphigenous, geniculate or straight, length variable, between septa 17-28 × 3-6 µm. Conidia acrogenous, borne singly or in chains of 2-4, smooth, irregularly ovoid, both ends rounded, or ellipsoid, or conical-ellipsoid, gradually tapering into a beak; beak (a secondary conidiophore) concolorous with the main conidial body, straight, 20-37 × 3-7 µm. Spore body pale brown to dark olive-buff, becoming darker with age, verrucose, transverse septa 1-10, longitudinal septa 0-5, constricted at septa, varying in size; length including beak 15-89 µm, width 7-30 µm

Host range: *Triticum aestivum* L.

Geographic distribution: Southern and southwestern Asia.

Notes: Simmons (1981, 2007) provides a more detailed description based on the type specimen and cultures derived from it. The size of conidia is greatly influenced by the medium on which the fungus is grown. The conidia range from 17-85 × 7-29, 15.5-68 × 7-13.5 and 18.5-88 × 7-17 µm on malt extract agar, standard nutrient agar and the host, respectively (Prabhu and Prasada, 1970). On malt extract agar the aerial mycelium is fluffy to cottony, 1-3 mm high, deep olive grey to dark olive grey; the surface of the culture is rough and tufty. Submerged mycelium is radiate to branching, olivaceous-black, producing a colony 80-90 mm diameter at 25°C. On standard nutrient agar, mycelium is velvety and the conidiophores and conidia form a velvety buff-olive cover. The colony surface is rather smooth to rough with characteristic zonation. The submerged mycelium is dense and dark greyish-olive; the colony is 50-60 mm diameter after 10 days culture in the dark at 25°C. Standard nutrient agar is a suitable medium for the comparison of different isolates since the spores are more uniform in size, although smaller than those on the host (Prabhu and Prasada, 1970). Colonies on potato dextrose agar are discrete or effuse, dark blackish-brown to black, 80 mm diameter in 5 days with smooth and entire margins (Anahosur, 1978).

Prasada & Prabhu (1962) distinguished the species as an *Alternaria* producing short chains of spores and having specificity on wheat. Other *Alternaria* species occur on wheat (Dugan and Peever, 2002). The identification of *Alternaria* species by examining morphological characters is not a simple task, requiring, at best, culture on specific media for the production of patterns of growth and sporulation, conidiophore architecture and conidium size, shape and surface ornamentation (Dugan and Peever, 2002; Simmons, 2007). Utilizing these criteria, Simmons places this fungus morphologically in a group with *A. infectoria*, a species often found on wheat, barley and other cereals (Simmons, 2007), but not a significant pathogen (Wiese, 1987; CPC, 2007).

There were some suggestions that *A. triticina* was an ecotype of *A. alternata* (Garg et al., 1972; Kumar and Rao, 1980; Rotem, 1994), but molecular studies clearly distinguish the two species (Pryor and Bigelow, 2003; Mercado Vergnes et al., 2006). *Alternaria alternata*, a common saprophytic species, sometimes referred to as *A. tenuis*, has been found in lesions with *A. triticina* (Prabhu and Prasada, 1966; Chaudhuri et al., 1976). Rao (1969) suggested that the leaf blight pathogen might be a variety of *A. tenuissima*, also common in India. Simmons (2007) clearly distinguishes among the three species on a morphological basis and noted that most of the wheat leaves received as specimens worldwide bear *A. alternata*, *A. tenuissima*, or an *Alternaria* in the 'infectoria complex', rather than *A. triticina*. Mercado Vergnes et al. (2006) observed the morphological differences described by Simmons as well and found some isolates from wheat reported as *A. triticina* to be nonpathogenic *A. alternata*, *A. tenuissima* or *A. arborescens*.

Molecular studies show *A. triticina* to belong genetically in the 'infectoria species group; that is basal to an *Alternaria/Ulocladium/Nimbya/Embellisia* evolutionary clade of hyphomycetes (Pryor and Bigelow, 2003; Xue and Zhang, 2007; Andersen et al., 2009). Pryor and Bigelow (2003) suggest that proper phylogeny-based taxonomy would require either redefinition of the genus *Alternaria* to include the other hyphomycete genera in the larger clade or redefinition of the various genera represented by the subclades. The second resolution could result in a new anamorph genus created for the *A. infectoria* species group, including *A. triticina*. Results obtained by Xue and Zhang (2007) place *A. triticina* close to *A. oregonensis*, another species isolated from wheat that belongs to the 'infectoria species-group' (Dugan and Peever, 2002; Simmons, 2007). This species group is connected through several of its members to the teleomorph genus *Lewia* in the Pleosporaceae (Simmons, 1986; Kwasna and Kosiak, 2002).

DISTRIBUTION

The occurrence of a blight disease caused by an *Alternaria* species on wheat was first recorded from Bihar, Uttar Pradesh, and Andhra Pradesh, India. The disease is now known to occur in most wheat-growing states of India (Prabhu and Prasada, 1970; Nema, 1986; Chowdhury and Roy, 1995; Sharma et al., 1998) where it is considered one of the most important foliar diseases of wheat. In addition, the fungus has been reported from North Africa (Anahosur, 1978), Mexico (Waller, 1981), Bangladesh (Rashid et al., 1985; Ahmed et al., 1994), Italy (Frisullo, 1982; Casulli, 1990), the Middle East (Wiese, 1987), Nigeria (Waller, 1981), Lebanon (Logrieco et al., 1990), France (Logrieco et al., 1990), Greece (Logrieco et al., 1990), Macedonia (the former Yugoslav republic) (Logrieco et al., 1990), Portugal (Logrieco et al., 1990), Egypt (Beshir, 1994), Turkey (Ozcelik and Ozcelik, 1997), China (Guo, 2005) and Argentina (Perello and Sisterna, 2006). A fungus identified as *A. triticina* has been isolated from crown rot of banana in Australia (Jones, 1991).

There is difficulty in identifying species in this genus, despite recent evidence by morphological studies (Dugan and Peever, 2002) and molecular methods (Mercado Vergnes et al., 2006) of a distinction of this species from some others frequently found on wheat leaves. Simmons (2007) states that the only correctly identified specimens of *A. triticina* he observed came from India and Iran. Mercado Vergnes et al. (2006) determined that some presumed *A. triticina* isolates were in fact those of other species, and stated the need for pathogenicity tests to confirm reports of the fungus from Germany, Morocco, Mexico, and Italy. Their tests showed only Indian isolates, and only some of those, to be *A. triticina* and pathogenic to wheat. Therefore, other reports from widely separated areas of the world may be equally in need of confirmation. However, Perello and Sisterna (2006) did establish pathogenicity by inoculation in their identification of *A. triticina* found on wheat in Argentina, as Frisullo (1982) did for some isolates in southern Italy.

Similarities to other species/conditions

Alternaria species are not easily identified correctly using morphological characters. Many isolates of other saprophytic species found in lesions on wheat are incorrectly identified as *A. triticina* (Mercado Vergnes et al., 2006; Simmons, 2007).

Simmons (2007) lists eight other definite species in the genus occurring on wheat and three occurring on barley. However, according to his key, the *Alternaria* species that are most morphologically similar to *A. triticina* in culture occur on *Pyrus*. Among the species from wheat, Simmons (1994) considers *A. triticina* very distinctive in conidium morphology and in the small number of conidia produced in culture on one conidiophore. The conidia of *A. triticicola* Rao, also found on wheat in India, differ in their greater length, often more than 100 µm (Simmons, 2007), although Prabhu and Prasada (1970) did not consider the differences sufficient to delimit a separate species.

Peever and Dugan (2002) provide a key to *Alternaria* species on grasses. In their culture of *A. triticina* originating from Prasada and Prabhu's type, conidia were significantly longer and wider on potato-carrot agar (PCA) than other small-spored, chain-forming species from grasses, including species of the *A. infectoria* complex. Other leaf spot-causing fungi occur on the aerial parts of wheat; most of these produce dark pycnidia or perithecia in the lesions (Wiese, 1987). Isolation of the pathogen would be required if the pathogen cannot be identified with certainty from visible symptoms and fungal structures.

NOTES ON CROPS/OTHER PLANTS AFFECTED

The main hosts of *A. triticina* are bread wheat (*Triticum aestivum* L.) and durum wheat (*Triticum turgidum* L. subsp. *durum* (Desf.) Husn. = *T. durum* Desf.). Durum wheats are more severely attacked by the pathogen than are bread wheats (Prasada and Prabhu 1962; Prabhu and Prasada, 1966; Singh et al., 1990). Nevertheless, the results of inoculations by Mercado Vergnes et al. (2006) lead them to consider this fungus a weak pathogen of wheat with little virulence on modern wheat cultivars.

The pathogen has also been reported to infect *T. dicoccum* (Kulshrestha and Rao, 1976), *T. sphaerococcum* (Kumar et al., 1974a), *triticales* (Chaudhuri et al., 1976; Wiese, 1987), barley (Mehiar et

al., 1976), oats (Logrieco et al., 1990) and rye (Logrieco et al., 1990). Prabhu and Prasada (1966), on the other hand, were not able to obtain infection by inoculation of barley, oats or ten species of wild grasses. Isolation from banana was reported, although the fungus was probably a secondary invader (Jones, 1991).

SYMPTOMS

The disease appears when wheat plants are 7-8 weeks old and becomes severe when the crop is mature. Infection is first evident as small, oval, discoloured lesions irregularly scattered on the leaves. As the lesions enlarge they become dark brown to grey and irregular in shape. Some are surrounded by a bright-yellow marginal zone. The lesions vary in size, reaching a diameter of 1 cm or more.

As the disease progresses, several lesions coalesce to cover large areas, resulting in the death of the entire leaf. In some cases the leaf starts drying up from the tip, prematurely, when lesions appear. Black powdery conidia may cover the lesions at this stage under moist conditions. The lowermost leaves are the first to show the signs of infection; the fungus gradually spreads to the upper leaves. In severe cases, similar symptoms are produced on the leaf sheath and stem, as well as the awns and glumes if spikes are infected at the pre-anthesis stage. If the spike is infected this early, seeds do not form. Infection at the dough stage of seed development results in glume infection, ear infection and seed infection. Heavily infected fields present a burnt appearance and can be identified from a distance (Prasada and Prabhu, 1962; Prabhu and Prasada, 1966; Singh, 1990).

DETECTION AND INSPECTION METHODS

The disease can be detected in the field on the basis of symptoms on the leaf, leaf sheath, awns and glumes. Lesions will lack the dark pycnidia or perithecia produced by some other leaf-spotting fungi (Wiese, 1987). Under severe conditions the heavily infected fields present a distinct burnt appearance that can be seen from a distance (Prabhu and Prasada, 1966).

The agar plate method is recommended for detection in seed (Mathur and Kongsdal, 2003).

DIAGNOSIS

Alternaria triticina may be isolated by plating of surface-sterilized infected plant parts. Pure cultures should be transferred to potato-carrot agar for culture and observation using the methods and keys of Simmons (2007).

Mercado Vergnes et al. (2006) identified differences in DNA sequences and PCR product sizes distinguishing *A. triticina* from other *Alternaria* species isolated from wheat leaves. Toxicity of culture filtrates to seedlings also was restricted to *A. triticina*. Their sequences of ITS regions of rDNA for the three *A. triticina* isolates are available on GenBank (NCBI, 2009).

PATHOGENICITY TEST

Cultures of the fungus are multiplied on standard nutrient agar (SNA) (1.36 g K₂HPO₄, 1.06 g Na₂CO₃, 5 g MgSO₄·7H₂O, 5 g dextrose, 1 g asparagine and 20 g agar in 1 litre distilled water) which is the best for sporulation (Prabhu and Prasada, 1966). For inoculation, a thick conidial suspension in water is sprayed on the plants. The inoculated plants are incubated in a humid chamber for 72 h before transfer to the greenhouse. Symptoms of infection appear 4 to 5 days after inoculation in the form of localized chlorotic streaks. The time required for sporulation varies with the prevailing temperature and humidity. Under high humidity, conidiophores will emerge through the stomata singly or in bundles of 2 to 10 (Prabhu and Prasada, 1966). In pot tests, a concentration of 40,000 spores/ml has been found to be essential to produce the disease consistently on all the leaves (except the top leaf) of 5-week-old plants. Susceptibility increases with plant age, and disease intensity decreases from the base to the top of the plants. In a resistant cultivar only small specks appear on the leaves (Kumar and Rao, 1979b).

BIOLOGY AND ECOLOGY

Alternaria triticina is both soilborne and seedborne. However, the soilborne inoculum is susceptible to extremes of high temperature and under the conditions of a hot summer such as that of Rajasthan, India, it may not survive until the next planting of the crop (Kumar and Arya, 1973a; Kumar and Rao, 1979c). The fungus has been shown to survive only 2 months in infected wheat debris on the soil surface during the summer months compared with 4 months in buried debris (Kumar and Rao, 1979c). Therefore, seedborne inoculum plays a major role in perpetuation of the pathogen. The initial inoculum carried with the seed may be able to multiply in the soil; plants are then infected when they reach the susceptible three-leaf stage. Younger seedlings are not infected (Prabhu and Prasada, 1966). The leaves in contact with the soil may be infected directly or spores may be splashed by rain to the lowermost leaves, from which the pathogen gradually spreads to the upper leaves. The spores germinate on the leaf surface producing germ tubes and appressoria (Prabhu and Prasada, 1966; Kumar and Rao, 1979b). Penetration of the leaf occurs through both the upper and lower surfaces. Germ tubes usually penetrate directly

through the epidermis and seldom enter the leafthrough stomata . The mycelium is both intercellular and intracellular. The outer epidermal cells collapse first, followed by the mesophyll cells which collapse slightly in advance of the mycelium. The fungus does not invade the conducting vessels in the xylem and phloem (Prabhu and Prasada, 1966; Kumar and Rao, 1979b).

Seven- to 15- day old seedlings are not susceptible to infection but susceptibility increases with plant age (Prabhu and Prasada, 1966). Disease development is maximum on 10-week-old plants (at the boot-leaf stage) when the relative humidity is high and the average maximum temperature does not exceed 25°C. Successful infection by the pathogen requires at least 48 hr in a saturated atmosphere. Maximum lesion development occurs at 25°C (Prabhu and Prasada, 1966; Ram and Joshi, 1978a).

Increases in levels of nitrogen and phosphorus fertilizer are reported to significantly increase (Prabhu and Prasada, 1970; Ram and Joshi, 1981) or significantly decrease (Rashid et al. , 1985) the severity of the disease . Disease incidence was also reduced by automobile exhaust pollutants (Kumar and Charaya, 1996).

PHYSIOLOGY

Glucose asparagine medium supports growth of the fungus although sporulation is poor to fair. Growth and sporulation are best at 25°C, at pH 6 and in continuous darkness (Kumar and Arya, 1978). Asparagine and sucrose support higher growth among nitrogen and carbon sources, respectively, and sporulation is maximal on media which are neutral or alkaline, rather than acidic, after growth of the fungus (Singh and Tyagi, 1978). Magnesium sulfate supports the best growth of the fungus (Kumar and Rao, 1979a). The fungus has been shown to produce toxins after 21 days at pH 8 and 15-25°C (Ozcelik and Ozcelik, 1997).

Conidia germinate after 3 h in water at 24°C. All the cells of the spores can produce germ tubes. There is a broad temperature optimum; between 15°C and 27°C more than 90% of the spores germinated within 24 h. There was appreciable germination, close to 20%, of conidia at 5°C whereas 76% germinated at 10°C. The percentage of spore germination gradually decreased above 30°C. Maximum germination of spores occurred at 100% RH; at 90% RH or less, spores did not germinate. The optimum temperature for growth of the fungus on malt extract agar appeared to be 25°C. Growth decreased very rapidly in the range of 30 to 35°C. At 35°C sporulation was retarded and abnormal aerial mycelium was produced (Prabhu and Prasada, 1966).

Physiologic specialization in *A. triticina* has been demonstrated. Jain and Prabhu (1977) recorded the occurrence of chromogenic variants in the pathogen. A chromogenic isolate designated race B produced a dark pigment in culture. It was less virulent than the non-chromogenic isolate race A, producing fewer and larger lesions with more pronounced chlorotic zones (Jain and Prabhu, 1977). Sokhi and Joshi (1972) reported that at least two races exist, whereas Garg et al. (1972) characterized six races of the fungus on 15 differential hosts using 20 collections from Rajasthan, India.

A culture filtrate of *A. triticina* inhibited seed germination and seedling root elongation and induced symptoms on the leaves of wheat plants similar to those induced by the fungus (Kumar and Rao, 1979d). Diluted culture filtrates injected into wheat leaves caused necrotic lesions in both resistant and susceptible genotypes (Mercado Vergnes et al., 2006). Seedborne isolates identified as *A. triticina* did not produce mycotoxins commonly produced by strains of *A. alternata* from wheat seed (Logrieco et al., 1990) or did produce some of the toxins (Bilgrami et al., 1995).

MOVEMENT AND DISPERSAL

Natural dispersal: Conidia develop on lesions under humid conditions and are dispersed by wind (Kumar and Arya, 1973a, 1976; Wiese, 1987). The fungus can also survive between seasons in crop debris (Prabhu and Prasada, 1966; Kumar and Rao, 1979c) and may produce inoculum in the soil (Prabhu and Prasada, 1966).

Accidental introduction: Although the pathogen is seed-borne, no definite introduction to a new region by this means has been demonstrated.

SEEDBORNE ASPECTS

Alternaria triticina has been shown to be seed transmitted in wheat (Prabhu and Prasada, 1966, 1967; Kumar and Arya, 1973a, 1973b; Dash and Narain, 1989). The pathogen can survive as conidia on the seed surface or as mycelium inside the seed coat (Bhowmik, 1969; Kumar and Arya, 1973a). Disease on the heads at the dough stage of seed development results in infection of glumes, awns and seeds (Prabhu and Prasada, 1966). A correlation between adult plant susceptibility and seed infection has been observed by Sharma et al. (1983). The extent of infection in seed varies from cultivar to cultivar and an average as high as 12.2% has been reported (Prabhu and Prasada, 1967). It has been suggested that the pathogen may have been transmitted through infected seed in the Kalimpong hills of West Bengal,

India (Kasier and Aslam, 1994). It has also been isolated from wheat seeds in Bangladesh (Ahmed et al., 1994), from wheat harvested in France, Greece, Italy, Portugal, Turkey and Macedonia (Logrieco et al., 1990), from seeds of barley from Greece, Lebanon, Macedonia and Turkey (Logrieco et al., 1990), and from oat and rye seeds from Greece (Logrieco et al., 1990).

Effect on Seed Quality

Seeds infected with *A. triticina* are often shrivelled, with a brown discoloration of the seed surface (Prabhu and Prasada, 1967). Infected seeds are small with a 46-75% reduction in weight (Raut et al., 1983). Seeds that appear normal in colour have also been shown to harbour the pathogen (Agarwal et al., 1972; Logrieco et al., 1990). Germination rate and seedling height are reduced when seeds are inoculated with conidia (Dash and Narain, 1989).

Pathogen Transmission

Alternaria triticina has been shown to be both soilborne and seedborne. However, the soilborne inoculum may not play a role in the perpetuation of the fungus under very hot conditions (Kumar and Arya, 1973a; Kumar and Rao, 1979c). It is primarily perpetuated, then, as conidia on the seed surface and/or as mycelium inside the seed (Kumar and Arya, 1973a, 1976). The initial inoculum carried with the seed may be able to multiply on decaying vegetable matter to such an extent that the soil itself can become infested (Prabhu and Prasada, 1966). The pathogen has been shown to survive in seed for 10 months (Kumar and Rao, 1979c).

Seed Treatments

Fungicide and hot water treatments have been used to reduce seedborne inoculum. Seed treatment with iprodione (Raut et al., 1983), thiram (Raut et al., 1983), 2-methoxyethylmercury chloride (Raut et al., 1983) and phenylmercury acetate (Ahmad and Ahmad, 1985) has been shown to be effective in reducing the level of infection. Seed treatments have also been shown to improve seed germination (Raut et al., 1983). Pre-soaking of seed in water for 4 h, followed by hot water treatment at 52-54°C for 10 min is also effective, without adversely affecting seed viability (Prabhu and Prasada, 1970).

Seed Health Tests

An agar plate method has been suggested for the detection of *A. triticina* from wheat seeds. A number of media have been used for detection. Seedborne infection can be determined on standard nutrient agar (SNA). Seeds are surface sterilized for 5 min in a mild disinfectant and rinsed three times with sterilized water. Five seeds are plated on SNA in a Petri dish and incubated for 8 days at 25°C in darkness. The colonies of the fungus are olive-buff with a velvety surface growth of conidiophores and conidia. SNA medium is effective for sporulation because the formation of aerial mycelium is greatly reduced. Sporulation is abundant only in darkness; it does not occur in continuous light. Reduced sporulation occurs when colonies are exposed to diurnal light and darkness (Prabhu and Prasada, 1965, 1967; Agarwal 1970; Agarwal et al., 1993).

Potato sucrose agar medium (PSA) (200 g potato, 20 g sucrose, 15 g agar, 1 litre double distilled water) supplemented with 10 mg/l of streptomycin sulphate has also been used for detection of the fungus from seeds of wheat, barley, oat and rye. The seeds were incubated for 7 days under fluorescent and 'black' (ultraviolet) lamps (2700 Lux) on a 12 hr light/12 hr dark cycle. The colonies of the fungus identified as *A. triticina* produced a characteristic ochraceous yellow colour at the margin of the colony. The spores were large, 18-90 × 6-30 µm, in short chains of 3-4 on PSA (Logrieco et al., 1990).

Kumar and Arya (1973a) used a glucose-glutamic acid medium for the detection of *A. triticina*. Seeds were incubated on the medium for 10 days at 25±2°C. Neergaard (1977) also suggested an agar plate method, and a plating method is recommended by a standard manual on seed testing (Mathur and Kongsdal, 2003). Raut et al. (1983) used a blotter test for the detection of seedborne *A. triticina*. Spores carried on the surface of seeds can be collected for observation by a washing test (Prabhu and Prasada, 1967).

IMPACTS

Leaf blight has caused serious damage on wheat crops, particularly in eastern and central parts of India. It is now considered to be one of the most important foliar diseases of wheat grown in the major states of India (Nema, 1986). Mexican cultivars and their derivatives are relatively more susceptible. Disease incidence is higher in durum wheat compared with bread wheat (Prabhu and Prasada, 1966; Nema, 1986). Leaf blight caused heavy yield losses in the early 1960s in Kenphads cultivars that were developed in the Maharashtra state for resistance to rust. This resulted in the withdrawal of these cultivars from cultivation. During the 1963-64 wheat season, the outbreak of the disease was so severe at the Botanical Sub Station, Pusa, Bihar that not a single genetic stock under cultivation escaped the disease (Prasada and Prabhu, 1964).

In severe epidemics the yield losses may exceed 60% (Prabhu and Singh, 1974; Sokhi, 1974). A severe epidemic in a wheat crop grown on 300 ha in the Vidarbha region of India caused a 75% reduction in grain weight (Raut et al., 1983). The extent of damage depends upon the cultivar and the stage at which infection occurs. The disease can appear in severe forms in experimental plots under artificial epiphytotic conditions causing severe losses. Estimation of losses indicated that there can be almost total loss of yield in a highly susceptible cultivar such as NP 830 when plants are infected at booting stage (Chenulu et al., 1970). According to Sokhi and Joshi (1974), the loss may reach 35% if the flag leaf and the leaf below it are badly blighted at heading stage; damage is close to that caused by disease on all the leaves. Likewise, Ram and Joshi (1979) observed that when the disease appears at the flag leaf stage under artificial conditions of infection, the loss in number of grains was up to 26.4%. The ear-head length is considerably reduced, affecting the number of grains. Infection causes a significant decrease in growth parameters, especially in susceptible cultivars (Beshir, 1994). It decreases the number of grains per ear, 1000-grain weight and yield (Ram and Joshi, 1981).

MANAGEMENT

Introduction

The control of leaf blight is primarily accomplished through seed treatment, fungicidal sprays and use of resistant cultivars. For details on Seed Treatments, see the section on Seedborne Aspects of Disease.

Cultural Control

Use of tested clean seed will prevent introduction to areas where the pathogen is not already present. Burial of crop debris can enhance survival during hot summer seasons (Kumar and Rao, 1979c), so delayed tillage of fields could be beneficial.

Chemical Control

The disease has been controlled through use of foliar fungicides: mancozeb (Nene et al., 1971; Singh and Singh, 1971; Bhowmik, 1974; Agrawal et al., 1976; Ojha and Mehta, 1977; Ram and Joshi, 1977; Singh et al., 1979; Verma and Khanna, 1988), ziram (Tandon et al., 1967; Sankhla et al., 1972; Kumar and Arya, 1973b; Bhowmik, 1974; Ahmad and Ahmad, 1985), zineb (Mishra et al., 1970; Singh and Singh, 1971; Ekbote and More, 1976; Agrawal et al., 1976; Ram and Joshi, 1977; Ojha and Mehta, 1977; Singh et al., 1979; Mishra et al., 1989; Trimurthy et al., 1993), thiram (Mishra et al., 1970), fytolan (Ojha and Mehta, 1977; Ram and Joshi, 1978b), propineb (Verma and Khanna, 1988), chlorothalonil (Verma and Khanna, 1988) and nabam (Ekbote and More, 1976).

The severity of infection by *A. triticina* was reduced from a measure of 7.7 to one of 1.9, and yield increased by 6.05 quintals/ha as a result of seed treatment with phenylmercury acetate followed by three sprays of ziram on plants at 15-day intervals (Ahmad and Ahmad, 1985). The fungus has been shown to develop tolerance in culture to progressively increasing concentrations of mancozeb (Sankhla et al., 1970).

Host-Plant Resistance

Wheat genotypes differ in their degree of resistance or susceptibility to leaf blight infection (Prasada and Prabhu, 1964; Prabhu and Prasada, 1966; Sokhi et al., 1973; Kulshrestha and Rao, 1976; Singh et al., 1977; Singh and Tyagi, 1979; Ahmad and Singh, 1983; Serrone and Porta Puglia, 1985; Dasand Roy, 1989; Singh, 1990; Singh et al., 1992; Borkar and Patil, 1995; Chowdhury and Roy, 1995; Singh, 1995; Singh et al., 1995). Durum wheat and cultivars having durum derivatives are more susceptible than bread wheat cultivars (Prasada and Prabhu, 1962; Prabhu and Prasada, 1966) although the reverse has been observed (Serrone and Porta Puglia, 1985). Resistance has been identified in some cultivars of wheat but many cultivars and genotypes, including the dwarfed wheats, are susceptible. There is abundant material available for the development of wheat cultivars resistant to *A. triticina*. Some of the highly resistant strains are Hope (E 41), H-44 (E 48), Ceres (E 50), Thatcher (E 124), Gobo (E 569), Cometa Klein (E 671), Frondoso (E 771) and La Prevision (E 928) (Prabhu and Prasada, 1966). Of 70 cultivars later tested against *A. triticina*, none was immune but three cultivars, HP 1163, HD 1941 and M-134, were resistant (Ahmad and Singh, 1983). The older Indian wheat cultivars, namely NP 4, 52, 200, 809 and 824, were resistant to the disease whereas cultivars NP 710, 761, 988 and 790 were tolerant (Singh, 1990). Singh et al. (1977) reported that lines/cultivars Arnautka, E 6160 and K 7340 were immune whereas resistance was shown by E 8682, HB 384, HD2157, HS 74, HW 2449, K 401, K 899, K 7333 and VL 417. Cultivar HP 1209 has been shown to be resistant to *A. triticina* (Dasand Roy, 1989). Mercado Vergnes et al. (2006) found that, contrary to some reports, modern spring bread wheat genotypes from south Asia and Mexico were resistant to *A. triticina* at the four-leaf stage and at heading.

The physiology of infected leaves of resistant and susceptible cultivars has been studied. Following inoculation with *A. triticina*, the amount of sugar and starch decreased in both resistant and susceptible cultivars. Phenol content increased in resistant leaves and decreased in susceptible ones. Free amino

acids, especially those involved in aromatic metabolism, increased markedly in resistant cultivars. The chlorophyll content decreased with infection. In resistant cultivars, disease progress ceased after 5 days and changes in phenol and nitrogen content continued for up to 10 days (Kumar and Rao, 1980). Resistance may be related to specific compounds in or on leaves. Susceptible cultivars contain larger amounts of sugars than resistant cultivars (Kumar et al., 1974a; Kulshrestha and Rao, 1977). There are also more phenolic compounds in resistant cultivars. Phenolic compounds accumulated more rapidly in resistant cultivars than in susceptible cultivars as a result of infection (Beshir, 1994). The physiochemical properties of the leaf surface are partly governed by the presence of wax. Kumar (1974) reported quantitative variations in wax content of wheat leaf surfaces: it was high in NP 4, a resistant cultivar, and low in the susceptible cultivar NP 83. Of the constituents, an acetate of n-w hydroxy acid was only found in the wax of resistant cultivars. The susceptible cultivar was characterized by the absence of n-w hydroxy acid, n-secondary alcohol and an unidentified lipid. Cuticle thickness, on the other hand, had no definite relationship with the resistance or susceptibility of wheat cultivars (Kulshrestha and Rao, 1978). Field studies with 90 genotypes suggested that a spreading plant habit might be preferred for selecting genotypes with resistance to *A. triticina*. A high negative rank correlation ($r = -0.8$) between plant habit and disease resistance was estimated (Singh et al., 1992).

On highly resistant *Triticum sphaerococcum*, *A. triticina* failed to penetrate the leaves; on moderately resistant cultivars germ tubes grew and branched but penetration did not occur. In susceptible cultivars, appressorium formation and penetration took place. In tissue cultures of the highly susceptible cultivar, almost all conidia in contact with the callus germinated and germ tubes directly penetrated the cells. Mycelial growth was intercellular and intracellular (Kumar et al., 1974b).

The basis of inheritance is not clearly indicated, but resistance is generally a recessive trait. A different recessive gene governed resistance to *A. triticina* in each of two resistant cultivars tested by Srivastava et al. (1981). Kulshrestha and Rao (1976) found that susceptibility of NP 830 was controlled by a dominant gene and that of NP 891 by two dominant complementary genes. Sokhi et al. (1973) reported that resistance to *A. triticina* was governed by a pair of recessive genes in Agra Local, NP 52, NP 54, by two pairs in NP 824 and NP 809, and by three pairs of dominant genes in E 1912. The field resistance of P 32-2-4 to *A. triticina* was recessive. Susceptibility was due to one dominant gene or two independently inherited dominant genes (Narula, 1982). However, none of 105 wheat genotypes tested by inoculation showed evidence that the two recessive duplicate genes confer complete resistance to *A. triticina* and *Cochliobolus sativus* in wheat (Singh et al., 1997). Additive components play a major role but dominance components also contribute significantly in controlling the variability for leaf blight resistance in wheat crosses. It is desirable to follow a simple recurrent selection scheme for higher tolerance in order to identify resistant plants among the segregating populations derived from crosses of parents of diverse origin following the pedigree method of breeding (Sinha et al., 1991).

GAPS IN KNOWLEDGE/RESEARCH NEEDS

As Mercado Vergnes et al. (2006) suggest, reports of *A. triticina* occurrence in areas distant from India should be confirmed by pathogenicity tests of the isolates on the apparent wheat hosts. In addition, molecular and morphological comparisons of the different species in the *A. infectoria* species-group reported from wheat in limited areas should be made to clarify the relationships and the distributions of the species that are pathogenic to wheat.

References

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